To summarize the ideas above expressed, in so far as they bear upon the linitis plastica, we arrive at the conclusion that this disease is not a new kind of morbid process, but is the effect of some chronic infective agent whose nature has not yet been determined. The process at work is often akin to the granulomatous process which occurs in mediastinal tissues and elsewhere, it is akin to the changes which occur in the course of some forms of tuberculosis and of diffuse sclerotic types of tertiary syphilis, and it is occasionally followed by the development of an epithelial growth, just as is sometimes noticed as a sequel to tuberculosis and syphilitic lesions.

The disease known as linitis plastica, or cirrhosis of the stomach, only demands a separate name because it is clinically a simulator of true carcinoma. Its prognosis should be more favorable and its treatment by the surgeon more effective.

It is further worthy of mention that the small degree of epithelial overgrowth which may be regarded by some observers as carcinomatous, in Cases II, III, and IV of our series, are not really any greater in extent than are the atypical proliferations of epithelium which are known to result from the subcutaneous introduction of certain irritant lipoid soluble bodies. The suggestion is that the abnormal cells in this granulomatous tissue are pouring out substances which may excite the overgrowth of the glandular epithelial cells in the same way that many bodies (dimethylamidoazobenzol, amidoazotoluol, α-naphthylamine, methylimidazol, indol, and skatol, etc.) were found to excite epithelial overgrowth by Wacker and Schmincke[12] in 1911. This renders the development of carcinoma as a secondary process on a preëxisting (often long-persistent) submucosal disease quite intelligible, and emphasizes the view which we would point out that the malignant change in this, as in "Hodgkin's disease," and in many lymphosarcomas (also in Paget's disease of the bones), is not an inevitable sequence, but is superimposed by the succumbing of certain other tissues to the continued noxious action of abnormal cell-products.

THE ALIMENTARY HYPERSECRETION OF CHRONIC ULCER AS SHOWN BY THE LACTOSE TEST MEAL.[1]

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Considerable data must be collected before the causation of anomalies of gastric secretion will be entirely understood. Much of the literature on the subject was written when clinical pictures were differently interpreted from the way they are today. The relation between stomach symptoms and diseases of other organs was little appreciated previous to the present decade. Chronic peptic ulcer was an almost unknown disease. Methods of diagnosis have gradually been improved, and entirely new methods have been originated.

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[12] Münch. med. Woch., 1911, p. 1607.

[1] Read before the American Gastro-enterological Association, June 3, 1912.

The literature concerning gastric secretion following the ingestion of test meals is meager and unsatisfactory. With the ordinary test-meals in vogue it has been impossible to gain any notion of the constitution of extracted chyme. As a routine, we have been most concerned with the presence and percentage of free acid and the total acidity. Sahli and Friedman have each proposed a test-meal containing fat which was to be used as an index of the amount of the chyme made up of test-meal residue. Little clinical work with these methods has been reported.

Last year at the meeting of the American Gastro-enterological Association, I urged the advantages of a test-meal in which lactose was used as an indicator. Let me briefly recall the composition of this meal and its method of use, referring to the original article[2] for necessary details. The test meal consists of 300 c.c. of weak tea or water to which is added 30 grams of lactose. With this the patient takes two unsalted, unsweetened crackers. At the end of an hour a portion of chyme is extracted, and then a definite amount of water, for example, 150 c.c., is injected through the tube and mixed with the chyme by partially withdrawing it and forcing it in again. From the difference in the acidity of these two samples the total stomach content is calculated by the formula suggested by Mathieu. The lactose percentage is then accurately determined in the first or undiluted specimen, and from that we determine the amount of sugar remaining in the stomach, and calculate the amount of the chyme which is made up of test-meal residue, and that which is made of gastric secretion.

During the last year I have continued the daily use of this test-meal, and have been convinced of its accuracy and the importance of the obtained data. Repeated tests in the same individual show that the results are consistent. Its accuracy in the estimation of the motor power of the stomach has been corroborated by Röntgenoscopy when used as an aid to diagnosis. In no thoroughly studied case have we been able to contradict that it gives an accurate idea of the motor power of the stomach for fluids. The question arises as to the accuracy of estimation of gastric juice secretion. Unfortunately, we are lacking in practical methods to verify the results, but from our studies we have no reason to question their accuracy.

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[2] Jour. Amer. Med. Assoc., March 16, 1912, lviii, 753.

We know how much test-meal has passed out of the stomach from what remains, and the question remains as to whether a proportionate amount of gastric juice has passed over. This problem is difficult of solution, as we have no means of knowing that in the hour of the test conditions are at all times the same as when an hour has expired. In the same case, tests taken at periods after the ingestion of the test-meal, varying from ten minutes to one hour, indicate that gastric secretion begins promptly after this test-meal; free acid is soon present, and even in ten minutes the stomach may contain from 50 to 100 c.c. of gastric juice. If it be true that the rate of gastric juice secreted and passed from the stomach corresponds proportionately with the test meal evacuated, there must be wide deviations in the amount of secretion, and, in certain cases, the amounts must be very great. This is a matter of much practical interest.

An idea of the amount of secretion is gained by the ratio which obtains between the test-meal residue and the gastric-juice residue. Thus, if we find 50 c.c. of test meal and 100 c.c. of gastric juice in the stomach, the ratio is 1 to 2. If the rate of exit of the two is constant, then, using the same example, the amount of gastric juice passed out would be 250 times 2 or 500 c.c. of secretion passed and 100 remaining. These figures are not surprising when we stop to think how large is the amount of gastric juice produced by a fractional part of the canine stomach, studied with the Pawlow method.

In order that we may appreciate the results obtained in chronic ulcer, it will be necessary first for us to fix in mind the figures for cases which have been selected as normal and those where certain definite conditions have been discovered other than the one under consideration. A large number of cases have been studied in search of possible gastric derangements and for statistical purpose. From these I have selected 10 which appeared to be within the normal limits of motility and secretion. Not every case, experimentally studied, proved to be as normal as its freedom from complaints might seem to indicate. This is, it seems to me, a thing we should expect.

TABLE I.—Normal Cases.

Case.	Free acid.	Total acidity.	Test-meal residue.	Gastric-juice residue.	Ratio.
1	19	40	30	47	1 to 1.6
2	34	55	33	80	1 to 2.4
3	20	60	34	50	1 to 1.5
4	30	50	35	81	1 to 2.2
5	32	47	45	85	1 to 1.8
6	16	54	29	80	1 to 2.8
7	32	44	24	41	1 to 1.7
8	20	40	26	31	1 to 1.2
9	30	50	35	80	1 to 2.3
10	10	41	50	97	1 to 1.9

There is little use in prolonging this table for normal cases, as the standard is necessarily more or less arbitrary. I regard the above figures as within the normal limits. We may say that more than 50 c.c. of test-meal residue shows deficient motility, and that the ratio of test-meal residue to gastric-juice residue should be about 1 to 1½. If the two are equal, or the gastric juice less than the test meal, there is deficient secretion. I would not place the upper limits of this ratio with an equal degree of positiveness. The question presents itself as to whether symptoms or pathological digestion arise, as a rule, when the ratio reaches a certain figure. It will take a deal of study and clinical observation to determine such questions.

TABLE II.—Enteroptotic Cases.

Case.	Free acid.	Total acidity.	Test-meal residue.	Gastric-juice residue.	Ratio.
1	18	42	84	60	1.4 to 1.0
2	10	35	85	68	1.4 to 1.0
3	16	42	123	41	3.0 to 1.0
4	8	28	216	150	1.4 to 1.0
5	9	26	101	104	1.8 to 1.0
6	34	60	15	44	1.0 to 2.8
7	30	50	57	76	1.0 to 1.3
8	0	12	205	105	1.1 to 1.0
9	0	18	40	30	1.3 to 1.0
10	0	12	34	48	1.0 to 1.3
11	10	26	62	102	1.0 to 1.7
12	0	12	32	48	1.0 to 1.5
13	60	46	47	47	1.0 to 1.0

In order to give some idea what takes place in the general group of cases spoken of as enteroptotics we have compiled Table II. It is apparent that in the main these cases are characterized by some degree of motor insufficiency. In some cases the motor capacity for fluids is fair or even excellent. In quite a large proportion the secretion is low. In this type of case it rarely appears high. Case 8 in Table III is shown to have a fair degree of hypersecretion. In this case there was a marked enteroptosis.

TABLE III.—Chronic Appendicitis.

Case.	Free acid.	Total acidity.	Test-meal residue.	Gastric-juice residue.	Ratio.
1	22	41	75	105	1.0 to 1.4
2	60	76	25	86	1.0 to 3.2
3	12	32	85	60	1.4 to 1.0
4	20	44	75	106	1.0 to 1.4
5	34	60	59	103	1.0 to 1.7
6	50	65	51	120	1.0 to 2.3
7	20	35	28	84	1.0 to 3.0
8	44	64	43	75	1.0 to 1.7
9	12	30	101	80	1.2 to 1.0
10	0	18	40	33	1.2 to 1.0

These cases were all explored and the sole lesion was a chronic appendicitis. In Cases numbered 1, 6, 7, 8, there was epigastralgia of severe grade which ceased after appendectomy. From the marked difference in the degree of acidity and gastric juice in these cases it would seem doubtful that the acid secretion caused the pain, either directly or through inducing pylorospasm. In Cases 2 and 8 there was hunger pain relieved by alkalies and the ingestion of food. The high secretion in these cases may have been a factor in the causation of the discomfort.

Table IV.—Gall-bladder Diseases.

Case.	Free acid.	Total acidity.	Test-meal residue.	Gastric-juice residue.	Ratio.
1	30	50	80	90	1.0 to 1.1
2	44	72	31	67	1.0 to 2.2
3	28	40	211	142	1.6 to 1.0
4	0	15	65	65	1.0 to 1.0
5	0	13	112	53	2.0 to 1.0
6	38	51	57	84	1.0 to 1.4
7	8	32	33	44	1.0 to 1.3

Gallstones were found at operation in the first 3 cases. In the other 4 considerable observation has justified a positive diagnosis of gall-bladder trouble, but operation has as yet been refused. Test-meal studies have not been necessary in the majority of our cases, as the diagnosis was sufficiently clear to allow the recommendation of operation unhesitatingly. In the cases reported the nature of the complaints was such as to make differential diagnosis between a gall-bladder disease and other abnormal conditions somewhat difficult. The results of the test meal were of great value in reaching conclusions, particularly in Cases 1 and 3. Here there was epigastric hunger pain relieved by food. In Case 3 there was a hematemesis after prolonged vomiting, but exploration showed no ulceration.

Table V.—Ulcer Cases.

Case.	Free acid.	Total acidity.	Test-meal residue.	Gastric-juice residue.	Ratio.
1	56	80	36	149	1 to 4.0
2	66	80	34	136	1 to 4.0
3	46	68	55	224	1 to 4.0
4	44	65	20	150	1 to 7.0
5	36	50	37	130	1 to 3.7
6	46	60	64	212	1 to 3.4
7	80	120	15	138	1 to 9.0
8	38	50	38	119	1 to 3.2
9	36	61	10	94	1 to 9.4
10	20	44	36	139	1 to 3.7

It is obvious that in this group of cases there is a decided tendency to alimentary hypersecretion. (There is the question of the correctness of the diagnosis, sometimes admittedly a difficult matter.) Only the first 2 cases have been submitted to operation, and in both there was duodenal ulcer. In Cases 4 and 5 there could be no doubt of the diagnosis, as there was marked overnight stasis. The diagnosis in the other cases had been made on the history and *x*-ray findings, previous hemorrhage and occult bleeding when first studied.

To the above cases of ulcer 2 may be added upon whom gastro-enterostomy was performed before test-meal studies of this kind were instituted. Operation did not result in entire relief of symptoms.

TABLE VI.—Unsuccessful Gastro-enterostomies.

Case.	Free acid.	Total acidity.	Test-meal residue.	Gastric-juice residue.	Ratio.
11	16	32	38	124	1 to 3.2
12	17	35	11	69	1 to 6.0

The consistency of results in repeated observations on some of these ulcer cases is shown by the following figures where cases have been under medical treatment.

TABLE VII.

Case.	Test-meal residue.	Gastric-juice residue.	Ratio.
2	34	130	1 to 4.0
	49	120	1 to 2.4
	26	102	1 to 6.4

Benefit in subjective symptoms had apparently resulted when the second test meal was taken. An exacerbation of symptoms at the time of the third meal made operation advisable.

TABLE VIII.

Case.	Test-meal residue.	Gastric-juice residue.	Ratio.
3	55	224	1 to 4.0
	46	187	1 to 4.0
	21	113	1 to 6.0
	52	115	1 to 2.2
	55	137	1 to 2.3
	22	128	1 to 5.0
	41	71	1 to 1.8
	44	105	1 to 2.4
	42	118	1 to 2.7
	51	120	1 to 2.3

This case, under medical treatment, has suffered the usual remissions and exacerbations, refusing radical operative treatment. The test meals have been taken during the last year. For the last four months the patient has been free from symptoms, and during this period the last four meals were examined.

TABLE IX.

Case.	Test-meal residue.	Gastric-juice residue.	Ratio.
4	20	150	1 to 7.5
	22	178	1 to 8.0
	16	90	1 to 6.0
	40	110	1 to 3.0

This case is interesting in that it shows that although there is pyloric stenosis, as shown by large overnight stasis of rice and rasins, the fluid test-meal residue may be small. Operation was refused and the lowering of the secretion ratio followed diet, medication, and systematic lavage. There was marked improvement in subjective symptoms corresponding with the fall of gastric juice secretion.

CONCLUSIONS. The demonstration of an alimentary hypersecretion is strongly suggestive of a chronic gastric ulcer. Whether a low ratio occurs in a case of ulcer of this type must be determined by future observations. How frequently alimentary hypersecretion occurs in conditions other than ulcer cannot be stated on this comparatively small number of studied cases. In a few instances we have found the gastric juice three times the amount of the test-meal residue. A ratio as low as this has proved the exception in those cases in which ulcer has been diagnosticated.

INTERMITTENT SPINAL CLAUDICATION.

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INTERMITTENT claudication may be divided into (1) the cerebral type, (2) the peripheral type, (3) the lumbar type, and (4) the spinal type. The arteries most commonly involved are the tibial, brachial, coronary, mesenteric, cerebral, and spinal arteries.

The chief cause is arterial disease, arteriosclerosis, senile calcification, obliterating endarteritis, acute arteritis. Not only are there organic changes in the vessel walls, but also an associated vasomotor neurosis, with a tendency to vascular spasm, which narrows the lumen of the vessels and diminishes the flow of the blood to the parts, with a resulting ischemia. Oppenheim recognized a benign purely functional type due to vascular spasm. Venous changes or diseases may give rise to symptoms of claudication similar to those in case of arterial disease. A secondary change follows in the nerve trunks and fibers as would be expected from impaired nutrition.